United States
Environmental Protection Agency

AIR TOXICS MULTI-YEAR PLAN

Office of Research and Development




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The Office of Research and Development's (ORD) multi-year plans (MYPs) present ORD's proposed research (assuming constant funding) in a variety of areas over the next 5-8 years. The MYPs serve three principal purposes: to describe where our research programs are going, to present the significant outputs of the research, and to communicate our research plans within ORD and with others. Multi-year planning permits ORD to consider the strategic directions of the Agency and how research can evolve to best contribute to the Agency's mission of protecting human health and the environment.

MYPs are considered to be "living documents." ORD intends to update the MYPs on a regular basis to reflect the current state of the science, resource availability, and Agency priorities. ORD will update or modify future performance information contained within this planning document as needed. These documents will also be submitted for external peer review.

AIR TOXICS MULTI-YEAR PLAN

1.0 Introduction

The Office of Research and Development's (ORD) multi-year plans (MYPs) serve as a tool to plan the direction of its research program and to communicate that program within and outside ORD. This MYP will help ensure the relevance, quality and performance of ORD's air toxics research program which is intended to answer critical scientific questions that will reduce the uncertainty of risk assessments and produce more effective risk management practices for air toxics. In addition, the MYP provides logic, sequencing, and prioritization of ORD air toxics research such that the regulatory needs of the Office of Air and Radiation (OAR), Regions, states, and tribes can be met. This MYP is an extension of ORD's draft Air Toxics Research Strategy (ATRS) and is designed as a more detailed description of the direction established in the Strategy while also embodying the goals of the ORD and EPA Strategic Plans. The ATRS specifies the development of an MYP as one method by which the strategic direction of air toxics research will be implemented. This MYP seeks to answer key research questions established in the ATRS to support assessment and management of air toxics.

1.1 Relationship to Other MYPs

The Air Toxics MYP identifies the critical paths for resolving air toxic problems. Thus, this MYP is a problem driven MYP. The Clean Air Act Amendments of 1990 (CAAA) and other EPA regulatory programs identify the problems toward which air toxics research should be directed. Some air toxics research needs will also be resolved by findings from several other MYPs. Although the priorities for air toxics research are focused on human health, there are likely to be indirect ecological benefits (Ecosystem Protection MYP) to the extent the research planned leads OAR, Regions, and the states to adopt human health protection strategies that also reduce deposition to ecosystems. ORD's ecological research on air toxics stressors is also covered under the Water Quality, and Endocrine Disruptors MYPs. In addition, ORD participates in the Agency's program to address pollutants, including air toxics, that are persistent and bio-accumulate in the environment (PBT pollutants) such as dioxin and mercury. Of particular relevance to air toxics is the Mercury MYP. The goals in the Mercury MYP are to prevent and reduce the release of mercury into the environment and to improve the understanding of the environmental fate and transport of mercury. Research conducted under the Air Toxics MYP will support the goals of the Mercury MYP and vice versa. ORD's Pollution Prevention MYP includes research on innovative techniques to reduce and measure air toxic emissions from indoor, stationary and area sources. The Human Health Risk Assessment MYP supports development of generic risk assessment methods and tools that when completed can be applied to a variety of Agency programs including air toxics. The Contaminated Sites MYP includes work to complete several dose-response assessment documents for air toxics that are important to contaminated site assessment, e.g. trichloroethylene, polychlorinated biphenyls, and perchloroethylene. Particulate matter (PM) is a complex mixture made up of many different compounds, some of which are air toxics (e.g. metals, PAHs, and other components of diesel

exhaust). Thus, the Air Toxics MYP will derive benefit from PM research planned in the PM MYP. While no MYP exists for the Integrated Risk Information System (IRIS), a large amount of dose-response assessment work done in that program will also support air toxics needs. Similarly, Homeland Security areas of research need relevant to air toxics includes fate, transport, and infiltration; building decontamination; and rapid risk assessment.

1.2 Scope and Customer

The MYP addresses ORD planned research over the next eight years (FY03-FY10) while assuming the most recent President's Budget (*infra vide*, 1.3 Size of Annual Resources). Where appropriate, reference and links to other research outside ORD and EPA are made. ORD understands that priorities in air toxics research may change as assessments and controls are effected at national and urban scales, for residual risk determination, for mobile and indoor air sources, and at sites of concern to the Regions, states, and tribes.

The Air Toxics MYP describes the ORD's plans to support OAR's National Air Toxics Program under the Clean Air Act Amendments of 1990 (CAAA), the Mobile Source Air Toxics Strategy, and the Indoor Air Strategy as well as the needs of the Regions, states, and tribes. Some research will also be used primarily within ORD to improve our ability to support these groups in assessment and management of air toxics. Emerging research needs identified by ORD's clients include the ability to determine and control risk from acute exposures to air toxics, how to account for multipathway exposures to air toxics and the attendant risk, and tools that will permit community based risk assessments and control of air toxics. More specifically, air toxics research will support the following elements of the National Air Toxics Program and other needs of our clients:

- the National Scale Assessments which are performed every three years
- residual risk assessments for regulated stationary sources
- listing and delisting of hazardous air pollutants
- research in mobile source air toxics assessments and the Technical Analysis Plan, to support the development of mobile source regulations, e.g., standards for heavy duty vehicles, non-road spark ignition engines, and non-road compressions ignition engines
- research to support the development of an Indoor Air Strategy as well as the conduct of indoor air assessments
- community-based risk assessments and risk reduction projects. The Integrated Urban Air Toxics Strategy described the need for local risk assessments and risk reduction activities. There are a number of ongoing such projects, e.g., Cleveland pilot, and more are planned. Research supporting these risk assessments will be of benefit to OAR as well as Regional EPA offices, states and tribes.

ORD's air toxics research also benefits the Regions by providing the following:

- defendable IRIS toxicity values and exposure models for use in site specific works such as in the Superfund program and community assessments
- information on prioritizing pollutants and sources for enforcement programs and voluntary programs
- new and reliable techniques for Regions to use and promote in monitoring programs
- new and reliable techniques for Regions to use in measuring source emission and managing releases

1.3 Size of Annual Resources

The total dollars (extramural research and in house EPA personnel) for the air toxics budget has stayed constant at about $20 million since FY2000. This funding level includes an almost constant level of 90 FTE. The MYP is based on a constant funding level represented by the FY03 budget of $19.9 million and 88.8 FTE. Research area and administrative proportions of that FY03 budget are as shown in Figure 1a, and associated FTE are shown in Figure 1b. The air toxics budget has included grants under the Science to Achieve Results (STAR) Program and Congressional earmarks in the past, but no new STAR grants were awarded in FY00 through FY03. The grants amount shown is an earmark for the Health Effects Institute, but utilizes no FTE.

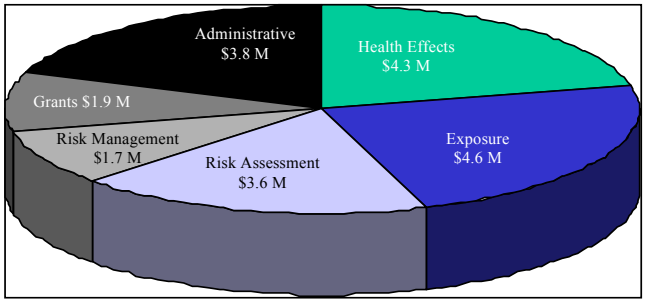

Figure 1a. Distribution of air toxics dollars per FY03 President's proposed budget.

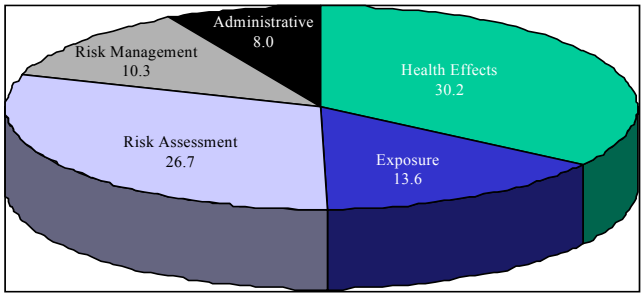

Figure 1b. Distribution of air toxics FTE resources per FY03 President's proposed budget.

2.0 Background

2.1 Key Regulatory Programs and Goals

The *National Air Toxics Program: Integrated Urban Strategy* was published July 19, 1999 and laid out the framework for EPA's risk-based air toxics program. This program is designed to

characterize, prioritize, and equitably address exposures to air toxics and their serious impact on the public health and the environment through a strategic combination of regulatory approaches, voluntary partnerships, ongoing research and assessments, and education and outreach. The program addresses emissions from large and small stationary sources, mobile sources and indoor air sources as part of its strategy for reducing risks from exposure to air toxics. In addition, the program prioritizes its actions and measures progress through the use of assessments conducted at multiple scales (e.g., national, regional, community).

There are four components to EPA's risk-based National Air Toxics Program: (1) source-specific and sector based standards, (2) regional and local initiatives which address multimedia and cumulative risks, (3) national air toxics assessments (NATA), and (4) education and outreach. The 3rd component, NATA, requires research to support all of the other components. It is the integrating piece of the National Air Toxics Program because it will inform and help prioritize efforts under the other three components, and is the vehicle EPA will use to track progress toward program goals. NATA will provide EPA and others with improved characterization of air toxics emissions, exposures, and risks for both stationary and mobile sources, as well as, risks from indoor air exposures. Eventually, as the science improves fully integrated assessments will address multimedia and multi-pathway exposures for single chemicals and chemical mixtures, and will address susceptible populations. The National Air Toxics Program will be one program by which EPA achieves its Clean Air Goal.

Under EPA's Strategic Architecture, the Clean Air Goal is to protect and improve the air so it is healthy to breathe and free of levels of pollutants that harm human health or the environment. This goal will be met by achieving major Objectives for Outdoor Air, Indoor Air, Atmospheric Change, Radiation, and Science/Research. This Air Toxics MYP does not directly consider Atmospheric Change or Radiation. The Outdoor Air Objective is stated as follows:

> **Objective 1.1: Outdoor Air**. Through 2010, and consistent with established schedules, emissions of outdoor air pollutants will continue to decline, and ambient air quality will improve to or be maintained at levels that protect public health and the environment....Healthy air for the other pollutants will be maintained for the 123.7 million people that had healthy air in 2001.

While omitted text in this Objective specifically cites criteria pollutants, the reference to "other pollutants" includes air toxics. EPA's expansion of knowledge about air toxics is described in three Sub-Objectives for outdoor air. The first Sub-Objective seeks to control stationary sources of air toxics by using market-based and other regulatory programs to reduce emissions. A key strategic target is that by 2007, federal air toxics regulations will reduce air toxics emissions by 2.2 million tons from their 1993 level of 3.7 million tons. making absolute emissions reductions in air toxics compared to 2000 levels. The second Sub-Objective intends to control mobile sources through federal regulations that will reduce air toxics emissions by 1.1 million tons from the 1996 level of 2.7 million tons. The third outdoor air Sub-Objective is directed toward reducing health risks and environmental effects from area source air toxics pollution found in localities including Indian country. The fourth outdoor air Sub-Objective is to reduce air toxics

risk at the local level by building on federally regulated emissions reductions. Targeted milestones are the 2004 public release of the revised National Air Toxics Assessment based on the 1999 inventory, development of an air toxics monitoring program, and the ability to characterize and assess trends for 20% of the Indian tribes in 2010.

The other objective relevant to air toxics under the Clean Air Goal is to achieve healthier indoor air:

> **Objective 1.2: Indoor Air.** By 2008, 4 million additional Americans than the 16 million in 2005 will be experiencing healthier indoor air in homes, schools, and office buildings.

Improving air quality in schools and workplaces will significantly depend on reducing exposure to air toxics. The goals are that by 2008 1,575,000 additional students and staff will experience improved air quality in their schools, and 720,000 additional office workers will experience improved air quality in their workplaces.

The regulatory objectives for outdoor and indoor air as stated above will require additional research in the area of air toxics. This research is described in Objective 1.5:

> **Objective 1.5: Science/Research** Through 2010, provide and apply a sound scientific foundation to EPA's goal of clean air by conducting leading-edge research and developing a better understanding and characterization of environmental outcomes under Goal 1.

Under this Objective, methods, models, data, and assessment research associated with air pollutants will be provided through 2010. The Air Pollution Research Sub-Objective is that air toxics research will develop and improve air quality models and source receptor tools, cost effective pollution prevention and other control options, and scientific information and tools to understand and characterize environmental outcomes associated with nationwide, urban, and residual air toxics risks. Coupling the success of the science/research and regulatory (outdoor and indoor) Objectives results in air that is healthy to breathe and free of levels of pollutants that harm human health or the environment as noted in the Air Toxics Logic Model (*infra vide*, Figure 2).

2.2 The Air Toxics Logic Model

An air toxics logic model (Figure 2) is useful in program planning because environmental problems (EPA goals) and decisions on how to regulate or otherwise resolve these problems are linked with research outputs and ORD resources.

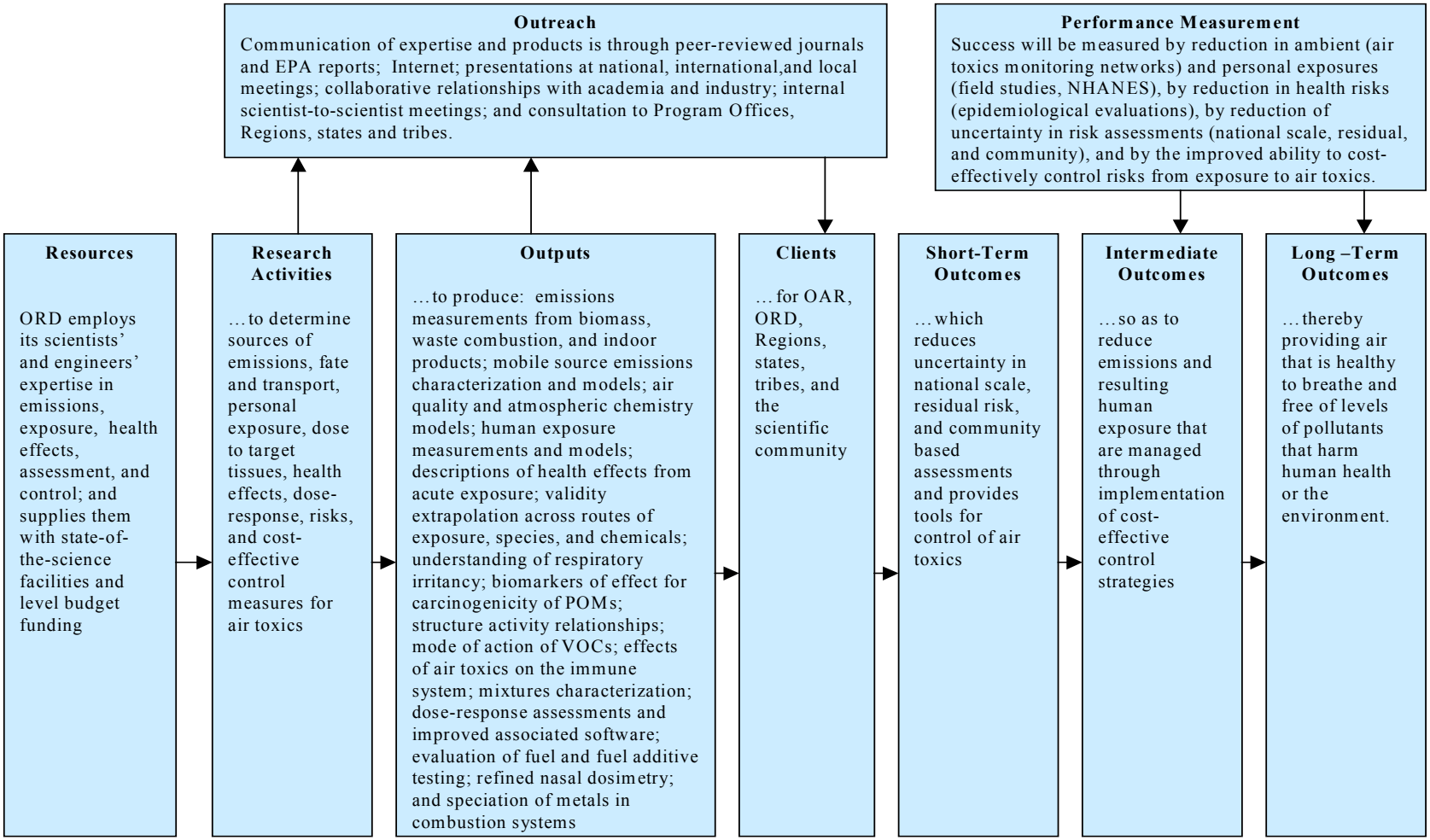

Figure 2. Air Toxics Logic Model

As stated in Goal 1, Clean Air, EPA's goal is for American communities and surrounding ecosystems to be safe from harmful levels of air pollution (Long Term Outcome). Safety from harmful levels of air pollution will be possible when risks associated with exposure are identified through scientific studies and sufficient information is available to characterize those risks in national scale, residual risk, and community scale risk assessments (Intermediate Outcomes). These assessments will include emissions from urban, stationary, mobile, and indoor air sources. The Long-Term Goals 1 and 2 in this MYP are the Short-Term Outcomes that will reduce uncertainty and control risks identified in these multi-scale assessments. Because the range of air toxics risk assessments is from national to community in scope, ORD air toxics research will be useful to clients in the Program and Regional Offices, states, and tribes. ORD's research outputs will include tools to characterize emissions; to model and measure exposure; to identify health hazards, mode of action and dose response relationships; to conduct dose-response assessments; to develop methods for risk characterization; and to investigate risk management options. This research will thus support the subobjective to develop tools for air quality modeling, pollution prevention, and risk assessment. ORD will use its scientists, engineers, and support staff (Resources) to conduct research activities on emissions; fate and transport; personal exposure; clinical, toxicological and epidemiological studies; and prevention and control technologies. The research program can be evaluated through indicators of accountability such as emissions inventories, risk assessment uncertainty, and cost-effective controls (Performance Measurement). Because communication of research results are important to the success of any program, these are also shown in the logic model (Outreach).

3.0 Long Term Goal Development

In order to achieve the Objective and Subobjective Goals for air toxics, the key science questions must be identified. From these key science questions long term goals for ORD research can then be developed.

3.1 Key Science Questions

There must be a focus to the key science questions that will resolve the greatest uncertainty in risk assessment and provide the most effective risk management. Both ORD and OAR have identified knowledge gaps that would reduce uncertainty in assessments to support the National Air Toxics Program. Some of these needs have been identified in *the Integrated Urban Strategy*, the *Final Rule on Control of Emissions of Hazardous Air Pollutants from Mobile Sources*, the *Residual Risk Report to Congress*, the SAB review of the *1996 National Scale Assessment,* and *Ranking Risks from Air Toxics Indoors.* An analysis of the identified knowledge gaps reveals research priorities for emissions, exposure, health effects, and assessment that, when completed, will reduce uncertainty in risk assessment and enhance the cost effectiveness of risk management. The following key science questions emerged from integrating the regulatory needs with our current knowledge on air toxics:

1. What are the rates and characteristics of air toxic emissions from indoor, mobile, and stationary sources and how do these emissions change based on various

operating conditions and other influences (e.g. mode of operation, building characteristics, process changes)?

2. What is the role of atmospheric transport, transformation, fate, and chemistry in air toxics concentrations (including indoor, micro-scale, urban, terrestrial, and regional concentrations)?
3. What is the relationship of concentrations of air toxics (from outdoor and indoor sources) to personal exposure?
4. What are the health hazards and dose-response relationships associated with exposure to air toxics
5. What improvements can be made to dose-response assessments to reduce uncertainty?
6. What health risks can be characterized quantitatively for people exposed to air toxics?
7. How can risks from air toxics can be prevented and managed cost effectively?

Collection of emissions data is critical to any air toxics assessment. Industry-specific emissions factors would help fill gaps or validate reported emissions used in inventories that form the basis of air toxics assessments. Emission factors for nonroad mobile sources also need to be determined. Mobile source emissions predicted from models need to be validated for both on-road and off-road sources. Methods accounting for indoor air emissions should be developed with an accounting for outdoor infiltration to air toxics and building usage. In addition, there is a continuing need for better techniques to measure air toxic emissions from all these sources including approaches that make continuous measurements.

Ambient concentrations of air toxics and the resulting actual human exposure to these pollutants need to be estimated with greater accuracy. To do this, air quality models which incorporate emissions, atmospheric chemistry (e.g. secondary formation of air toxics contaminants), and long range transport are needed. Evaluation of these models would be facilitated by a monitoring program which would also require additional monitoring methods for air toxics. Adaptation of these models to neighborhood scale will allow their use in community-level assessments and as a link to human exposure models. Human exposure models which estimate multi-pathway exposures and better characterize the uncertainty and variability in actual exposures are needed. To develop, evaluate, and refine these human exposure models, human exposure measurement studies are needed to estimate the relationship between ambient, indoor, and personal air toxics concentrations and to identify factors which influence exposures such as time spent in key microenvironments (e.g., vehicles, depots, residences etc.)

Dose-response assessments needed for risk assessments at multiple scales and from multiple sources are incomplete. An accounting of cancer and non-cancer dose-response assessments in the Integrated Risk Information System (IRIS) reveals many missing values for high-priority air toxics (see Appendix A, Table A-1). Many existing dose-response assessments have high uncertainty, have not been recently updated, or have not been externally peer-reviewed. An understanding of mode of action, respiratory irritancy, and target organ dose, including the development of physiologically-based pharmacokinetic models and shape of dose-response

curves will greatly facilitate the rapidity and increase the certainty with which non-cancer dose-response assessments can be developed. Being able to predict the shape of the dose-response curve for carcinogens will also increase certainty of the dose-response assessments for carcinogenic air toxics. Acute dose-response assessments should be developed and be based upon defined relationships between acute exposures and effects. Genotoxicity data and mechanisms will also help focus and speed development of dose-response assessment for carcinogens.

Effective risk management requires an understanding of emissions as well as knowledge about how specific aspects of a source influence emissions (e.g., operating conditions, pollutant speciation, plant design). Tools and data that assist Program Offices, Regions, and states evaluate and recommend risk management options for air toxic sources, particularly where residual risks will remain after MACT, are needed.

3.2 Long Term Goals

Two long-term goals to respond to the key science questions and which also serve as descriptors of the short-term outcomes within the logic diagram (Figure 2) have been developed. The first describes the tools ORD will provide to reduce uncertainty in risk assessments and the second addresses how ORD will aid in the assessment and cost-effective reduction of risk from exposure to air toxics.

- **LTG 1, Reduce Uncertainty in Air Toxics Risk Assessments**: By 2010, provide health hazard and exposure methods, data, and models to enable the Program and Regional Offices to reduce uncertainty in risk assessments of acute, chronic, and multi-pathway exposures to air toxics at the national and regional levels, and conduct 3-5 community-level exposure and epidemiology studies to characterize the risk of air toxics at that scale.

- **LTG 2, Implement Risk Reduction of Air Toxics**: By 2008, produce fifteen new or modified tools in the form of methods, models, or assessments that enable national, regional, state, or local officials to identify or implement cost-effective approaches to reduce risks from stationary point, area, mobile, or indoor sources of air toxics.

The LTGs function together as supporting elements of the risk assessment/risk management paradigm which is the process ORD uses to answer the science questions that will reduce risks from exposure to air toxics. Figure 3 shows the relationship of the two LTGs.

3.2.1 Value of Research for Reducing Uncertainty in Risk Assessment (LTG 1)

Under the National Air Toxics Program air toxics risk assessments will be conducted at multiple scales (national to community) and across multiple sources (stationary, mobile, indoor). The Science Advisory Board recognized the 1996 National Scale Assessment as containing too much uncertainty to be useful as a cost-benefit tool or a predictor of risk. Current residual risk

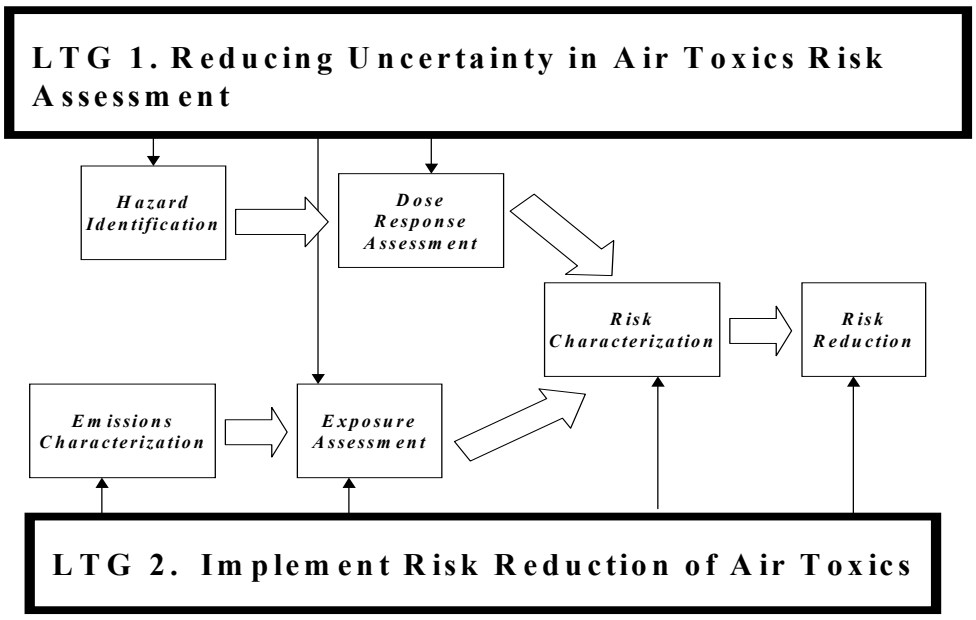

Figure 3. Relationship of Long Term Goals

assessments are also identified as containing significant uncertainties related to emissions, exposure, health effects, and dose-response. Community-scale risk assessments will require development of novel exposure and epidemiological concepts and methods before confidence in their findings can sufficiently inform the risk management process. As indicated in one of the strategic principles of the Draft Air Toxics Research Strategy, research and development in air toxics should be focused on the greatest risks to people and the environment. The research tools developed in LTG 1 are planned to reduce uncertainty in the many risk assessments conducted to identify risks to large populations whether at the national or community scale. There are several significant contributors of uncertainty in the assessment of risk from exposure to air toxics, including: the large number and variety of chemicals defined as air toxics; a lack of measurement methods and monitoring platforms; a lack of knowledge about their atmospheric chemistry; lack of data on the modes of action and dose-response relationships for both carcinogens and noncarcinogens; reliance on default assumptions used in dosimetry; and dose-response assessments. These uncertainties guide the selection and prioritization of research presented as annual performance goals (APGs) and annual performance measures (APMs) supporting LTG 1. Section 6.1 lists the APGs and APMs supporting LTG 1.

3.2.1.2 Logic in Narrowing Choices of APGs to Reduce Uncertainty in Risk Assessment

Figure 4 shows the critical paths (bold arrows) of APGs necessary for achieving LTG 1. The logic used to narrow choices of research to a critical path for achieving LTG 1 is based on developing science that yields the information necessary to apply the risk assessment paradigm as a solution to problems of air toxics risk. As shown in Figure 4, the critical path includes APGs that develop approaches for assessing the dose-response relationships for both acute and

chronic exposure to air toxics. Weaknesses in the 1996 National Scale Risk Assessment and ongoing residual risk assessments of stationary sources identify the need for developing methods to assess risk of acute exposure and for improving existing approaches to assess risks from chronic exposure. Research needs have also been identified in the overarching themes of mode of action, susceptible populations, and cumulative risk. Currently and over the near term, most research will address the need for developing tools to facilitate the risk assessment of individual air toxics in keeping with the current single-chemical approach to risk management. Intermediate-term research will include developing improved approaches to risk assessments for acute and chronic exposures. In the longer term, an increase in research regarding cumulative risk and susceptibility will occur. Because an emerging need is the ability to perform community scale assessments, a separate part of the critical path to LTG 1 is a preliminary effort which conducts pilot studies of community assessments as a proof of concept before final epidemiological and exposure tools are refined for use in larger such assessments. This research might largely be done through the Science to Achieve Results program of ORD. No one of these areas will exclude work in the other, but increased emphasis in emerging themes over time will reduce emphasis in research under previous themes.

3.2.2 Value of Research for Reducing Risk (LTG 2)

The results of the research conducted under LTG 2 are used directly to implement the risk reduction activities of EPA's air toxics program. The EPA program includes efforts to produce data and tools that can be used to develop and implement approaches that reduce risks associated with air toxics emitted from stationary and mobile sources in urban areas and air toxics found in indoor environments. Each of these elements of the program require the use of information and models to characterize risks and determine the appropriate EPA policy for management of risks. The research conducted under LTG 2 produces such information and models. Emissions data, new source and ambient measurement techniques, human exposure models, IRIS values, and risk management options are examples of information generated by ORD research and used by risk assessors and managers. Currently, the focus of the stationary source component of the EPA air toxics program is residual risk standards. These standards require EPA to assess the risk that remains after sources comply with MACT standards and tighten these standards when necessary. These assessments will benefit from ORD's research inputs. Mobile sources are a major contributor to air toxics emissions and exposures. The Clean Air Act authorizes EPA to periodically reassess the risks associated with mobile source air toxics and issue appropriate measures to reduce these risks as necessary. Future mobile source reassessments and actions to reduce air toxics emissions will benefit from ORD research inputs under this LTG. Program Office support for evaluation of toxicological testing of fuel and fuel additives (Clean Air Act, Section 211(b)) will be provided under this LTG. While there is no regulatory program for indoor air, to understand total exposures to air toxics, it is important to understand both the relative contributions of indoor and outdoor exposures and the relative contributions of outdoor and indoor sources to total human exposures. This information can be used in the development of voluntary and educational programs that inform the public about how to reduce risks from indoor air exposures to air toxics. ORD research under LTG 2 will play an important role in developing

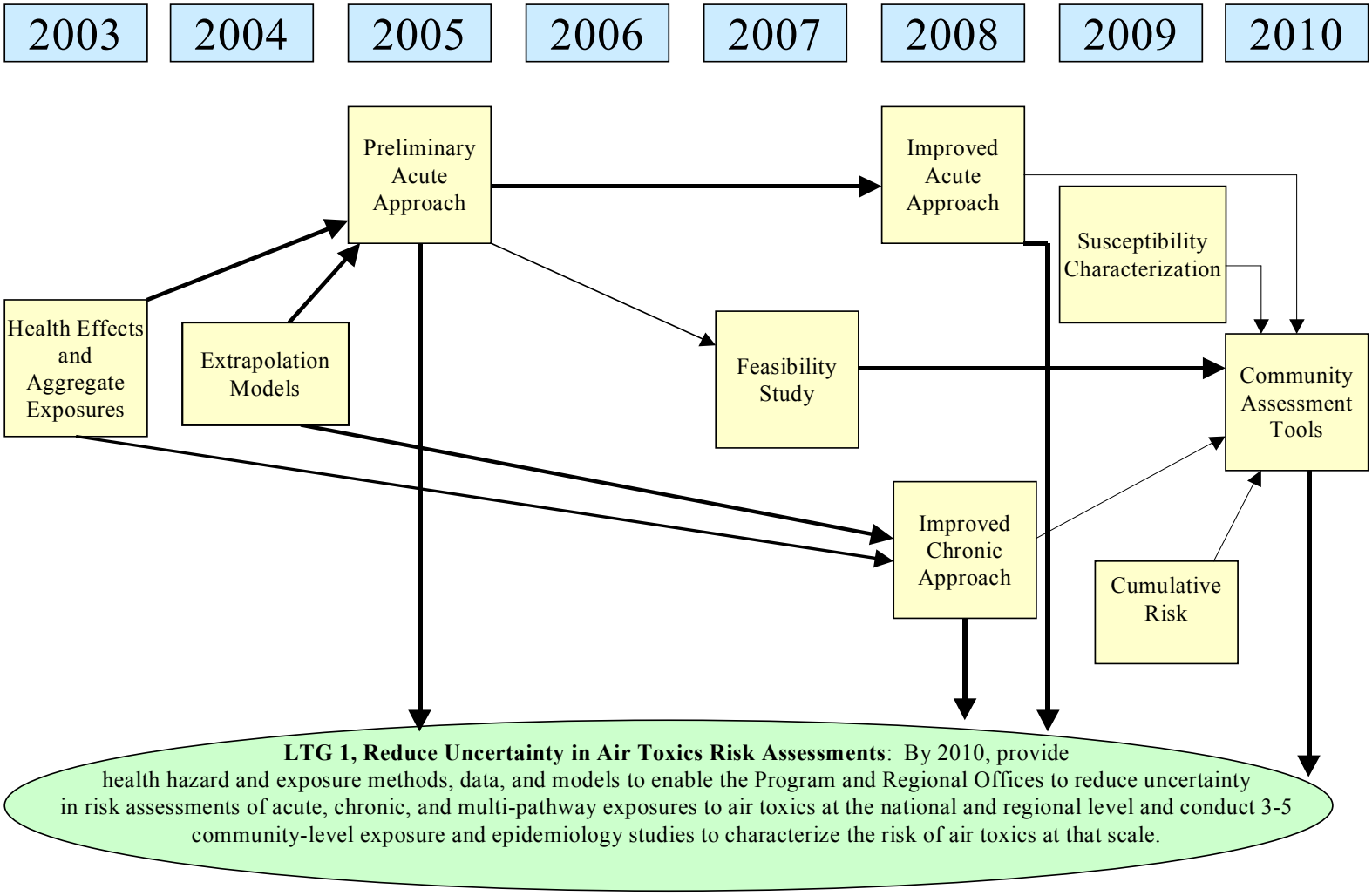

Figure 4 – Critical Path for Long-Term Goal 1

this information. Finally, EPA's air toxics program also includes efforts to reduce risk in urban locations. To measure progress in this area, the NATA activities include national scale assessments to characterize risks from all of the air toxics on the Urban Air Toxics list (see Appendix A, Table A-1) and from all of the sources emitting those air toxics. These national scale assessments use emission inventories, air quality and human exposure models, and health benchmarks to estimate risks. LTG 2 seeks to improve all of these assessment elements in order to provide risk managers with the best information available to support their decisions.

3.2.2.1 Logic in Narrowing Choices of APGs for Reducing Risk

Figure 5 shows the critical path for air toxics research under LTG 2. The bold arrows represent

critical paths; the lighter arrows are contributing paths; and the dotted arrows represent contributions from LTG 1 which are also shown in the table of LTG 2 APMs (section 6.2) in italics. The focus for research in LTG 2 is to enhance the programmatic activities of OAR. These programmatic activities include developing residual risk standards, assessing the need for and supporting development of mobile source air toxics regulations, communicating approaches to assess and manage risks from indoor air toxics and supporting efforts at local, regional, and national levels to identify and manage risks. LTG 2 includes APGs for each of these activities that are tied to the programmatic time line. For mobile source research, the goal of LTG 2 research is to provide information to support the assessment of risk from air toxics emissions. A mobile source air toxics rule was promulgated in 2000. When this rule was published, there were significant data gaps which needed to be addressed, so the rule contained a commitment to re-visit the provisions of the rule in 2004 and a technical analysis plan for filling data gaps. Some data gaps have been filled for the planned 2004 rule making, but ORD research will likely have the largest impact in the subsequent reassessment. While the date of this reassessment is not clear at this time, assuming another 4-years between reassessments leads to the 2008 APG which supports regulatory options for mobile sources. Research to support residual risk standards is needed on an ongoing basis. However, OAQPS is currently working on the "first" group of standards and will likely continue to do so in the near future. ORD can support the residual risk program by supplying information pertaining to the source categories that will be addressed in the future. LTG 2 includes two critical path APGs for supplying information for the 2nd and 3rd groups of residual risk standards in 2005 and 2008, respectively. ORD will need to coordinate with OAQPS to identify the sources of primary concern in each of the groups. OAR conducted the first national scale assessment of NATA in 2000 and is planning on conducting similar assessments in 2003 and every three years thereafter. Therefore, LTG 2 includes critical path APGs that provide information supporting these assessments in 2006 and 2009. The national scale assessments currently focus on the 33 chemicals on the Urban Air Toxics list. In order to prioritize research on specific chemicals ORD's air toxics research strategy (ATRS) includes four groups of air toxics. The first critical path APG in LTG 2 focuses on providing improved information for two of these groups (metals and aldehydes), and the second APG provides information for the remaining two groups of the ATRS (PAHs and halides). Finally, while no regulatory time line exists for indoor air activities, ORD's research is expected to produce significant information supporting these activities in the 2008 time frame. While each of these APGs support different

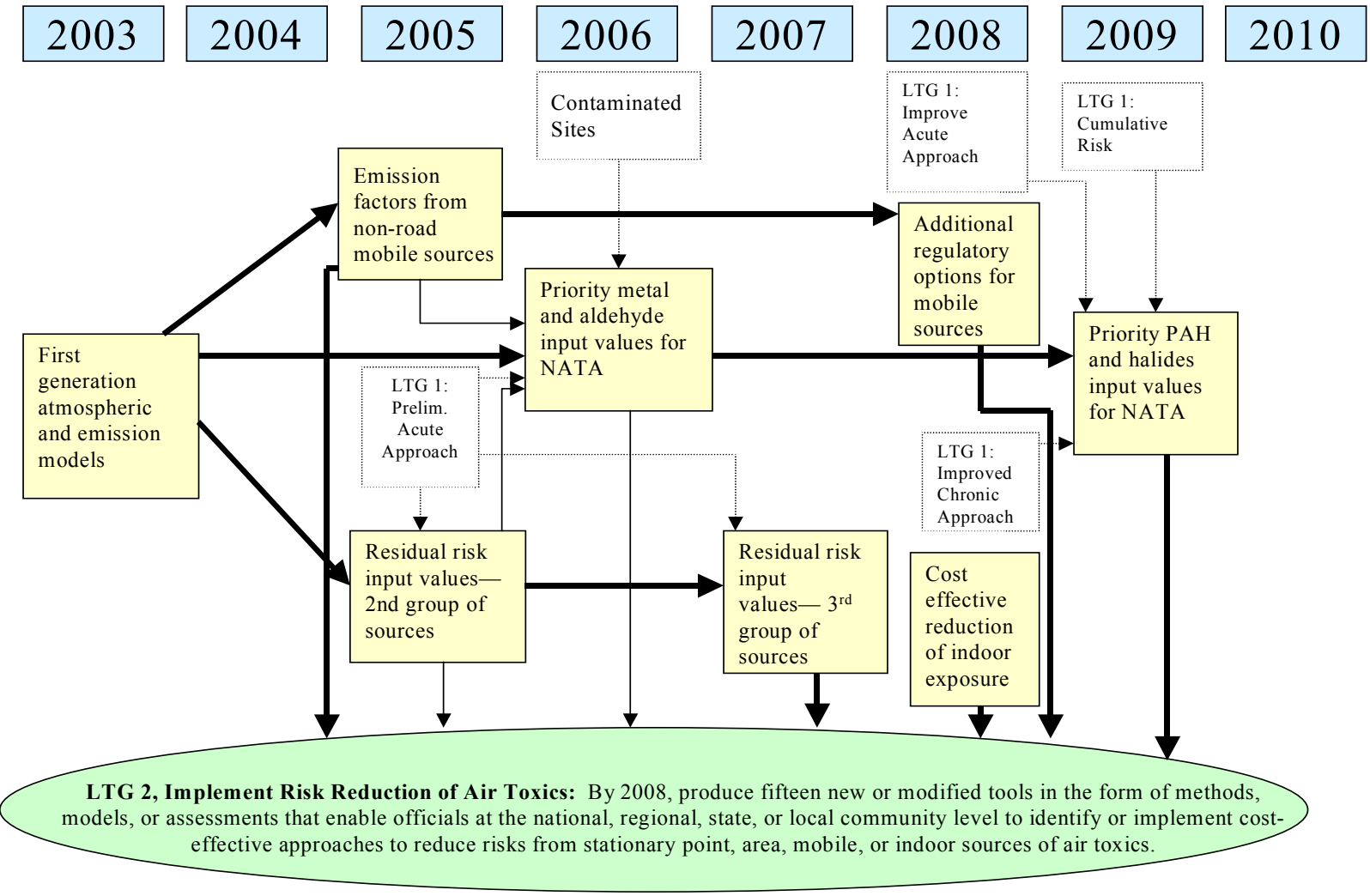

Figure 5 – Critical Path for Long-Term Goal 2

aspects of the EPA air toxics program, these APGs are also interrelated in that information and models produced to support one aspect (e.g., NATA) may also be applicable to other aspects (e.g., mobile sources or residual risk). This overlap can be seen in the priority list of air toxics for urban, stationary, mobile, and indoor sources in Appendix A, Table A-1.

3.2.3 Relative Emphasis of LTGs Over Time

The emphasis of air toxics research described throughout the time covered by this MYP is shown in Figure 6. The research under LTG 1 will emphasize the development of an acute approach to air toxics risk assessment in the early years (FY 2003 - FY 2005) and then improve this approach as well as the current chronic approach in the intermediate years (FY 2006-FY 2008).

Developing methods to effect community assessments will begin in the intermediate years and continue to the end of this planning period (FY 2007 - FY 2010). These efforts constitute a level emphasis of air toxics research from beginning to end of this plan.

The research under LTG 2 will emphasize the development of emission models for mobile sources and input values for quantifying and controlling residual risk in the early years (FY 2003 - FY 2005). Tools for supporting mobile source regulatory options, the next national scale assessment (3rd), quantifying and controlling the next group of residual risk sources, and developing cost effective controls for indoor air will be done in the intermediate years (FY 2006-FY 2008). Research to support the 4th national scale assessment will be provided through FY 2009. These efforts constitute a level emphasis of air toxics research from beginning to about 2008, and then a decrease.

Area	Emphasis in MYP Planning Window
LTG 1 - Reducing Uncertainty in Air Toxics Risk Assessment	Level then increasing for community assessments in 2009
LTG 2 - Implement Risk Reduction of Air Toxics	Level through 2008, and then decreasing

Figure 6. Table of Evolving LTG Emphases

4.0 Progress to Date

4.1 Research within ORD

ORD has made progress in air toxics research, and its accomplishments cover areas of emissions and exposure modeling, health effects, and dose-response assessment. The completed report "Emissions of Organic Air Toxics from Open Burning", which summarizes air toxics emissions data from open burning, assesses commonalities between combustion sources and develops methodologies for estimating the resulting emissions will support the Program Office's development of future emissions inventories and serve as a guide for future ORD research. ORD's Community Multiscale Air Quality (CMAQ) modeling system has been extended to predict ambient concentrations and deposition of some priority air toxics. Specific pollutants added to the modeling capabilities of CMAQ include formaldehyde, benzene, acetaldehyde, and acrolein. Research versions of the CMAQ model have been developed to predict ambient concentrations and deposition of mercury and dioxins (multimedia pollutants). The CMAQ model was also "exercised" by nested model runs at 36, 12, 4, and 1.33 km grids in Philadelphia, thereby demonstrating its ability to resolve exposure concentrations at neighborhood scales and be of particular benefit to OAR in local scale risk assessments.

ORD also outlined an approach to estimate human exposure, developed new information on health effects from high priority urban air toxics, and completed health assessments for the highest priority hazardous air pollutants to support national air toxics assessments. A "Research Plan for Air Toxics Human Exposure Measurement and Modeling" has been developed and peer reviewed. This plan will build upon previous efforts to characterize human exposure and the factors which influence these exposures including work funded via the STAR program. Health effects accomplishments include the discovery that some polycyclic aromatic hydrocarbons (PAHs) are metabolized to K-region dihydrodiols. These metabolites do not form DNA adducts, rather they cause DNA breaks and form quinones and reactive oxygen species. This novel mode-of-action (in contrast to Bay region epoxide formation) and mutation studies on benzo[a]pyene and dibenzo[a,h]pyrene will redefine the shape of the dose-response curve for some PAHs and reduce uncertainty in future dose-response assessments for PAHs. Similarly, the identified health effects of perchloroethylene have influenced development of the dose-response assessment for this priority air toxic. Cancer and non-cancer dose-response assessments for hydrogen sulfide, acrolein, ethylene dibromide, and methyl tertiary butyl ether have been prepared for external peer review. A report on the sufficiency of toxicity testing (Tier II) of baseline gasoline was provided with the result that additional testing is needed.

4.2 External Complementary Research

Because many of the general issues of concern to Air Toxics research in Goal 1 are also being addressed in the Human Health Research (HHR) program in Goal 8.2, the Air Toxics plan will benefit from significant products of research under Goal 8.2. These products include several projects under HHR FY 2008 APG "Identify pharmacokinetic/pharmacodynamic issues underlying uncertainties for extrapolation". The early APMs from this APG will inform the Air Toxics MYP APG 14 (2004) "Extrapolation Models", and later APMs from the HHR APG will inform both the Improved Chronic and Improved Acute APGs in the air toxics MYP. Other APMs from the HHR MYP will inform the Feasibility Study (e.g., methods to quantify health effects in children in epidemiological studies), Susceptibility Characterization (e.g., biological mechanisms of asthma, and age-related changes in susceptibility), and Cumulative Risk (e.g., methods and models of dose-additivity and response-additivity).

In addition, computational toxicology methods developed under Goal 8.2 may be useful for approaching complex issues in air toxics research. For example, the issues of genetic and age-related susceptibility, and the problem of potential interactions among the large number of HAPs may be amenable to study through computational toxicology methods. Finally, research products from the Air Toxics MYP will address questions of concern to other research plans in ORD. For example, the work on route-to-route extrapolation under the Extrapolation APG in the Air Toxics MYP will help inform the issue of aggregate risk in the HHR strategy.

Air toxics research is being conducted outside EPA also. The Center for Air Toxics Metals at the Energy and Environmental Research Center performs source characterization and risk mitigation research. The Mickey Leland National Urban Air Toxics Research Center contributes significantly to personal exposure approaches which will help reduce uncertainty in future risk

assessments at multiple scales. The focus of research at the Metropolitan Development Association of Syracuse and Central New York is assessment and mitigation of the impact of exposure to multiple indoor contaminants (including air toxics) on human health. The Health Effects Institute conducts research on the health effects of vehicle emissions. Health risk research involving some air toxics is also conducted at the Lovelace National Environmental Respiratory Center. The National Jewish Medical and Research Center focuses its air pollution research on the molecular pathogenesis of environmental lung disease, the biologic and genetic basis for individual susceptibility, and exposure/host response relationships.

More air toxics research is needed than ORD alone can provide. While this MYP is a plan of ORD research, ORD will endeavor to coordinate air toxics research needs with research organizations outside of EPA (e.g. other federal agencies, private sector groups, academia, and nonprofit research institutions). As an example, a critical need for conducting air toxics research is dose-response data, but the resources required to gather this data are not available in ORD. ORD will look to other sources to provide this data. The National Toxicology Program (NTP), which routinely conducts testing of carcinogens, might be one source of such data. ORD may exercise EPA's authority under the Toxics Substance Control Act (TSCA) to have chemical manufacturers provide this toxicity testing data.

5.0 Additional Research Desired

One of the National Air Toxics Program goals is to focus on multi-pathway and cumulative risks at the community level, where risks are least well characterized. Research is needed to reduce uncertainty in risk assessments of exposure to air toxics in urban communities and provide tools for controlling risks. ORD would expand its air toxics research agenda in the areas described below if the air toxics budget were increased by 10 to 20%. This research would involve the intramural and extramural programs and addressed sequentially as follows:

- Development of improved analytical methods for measuring ambient and personal exposure to high priority air toxics are needed–specifically, methods that improve temporal resolution, sensitivity and stability. The methods would also include the development of personal monitors and local stationary monitors that are smaller, less expensive, and more rugged than current monitors.

- Assessments of emissions and exposure to high priority air toxics in targeted microenvironments are needed. Unlike criteria pollutants which are more ubiquitous, air toxics may be a problem of hotspots or even specific microenvironments. Microenvironments include homes with attached garages, businesses, automobiles, residences near fence lines, etc.

- Epidemiological studies of health risks from exposure to air toxics in communities with significant exposure (hotspots) are needed. Because air toxics hotspots may represent significant risks, the assessment of these hotspots through epidemiological studies may provide the information needed to control risks in specific areas without national

regulations. A proven epidemiological approach to the assessment of hotspots is lacking in our current assessment approaches.

While ORD recognizes that the above broad knowledge gaps exist, there are also smaller, more specific research areas that would be fulfilled with more limited increases in resources. With the number of HAPs for which emissions, exposure, health effects, dose-response assessment, and control need to be addressed, the current program is unable to address all the needs in these areas. Below is a list of prioritized research projects, essentially aligned along the critical science questions, that would be funded if additional limited budget were available.

Emissions

1. Develop cannister/sorbent sampling for rapid air toxics analysis with Jet REMPI
2. Develop a mobile, trace organic open burning sampler ("Nomad" sampler)
3. Characterize major urban sources of low concentration VOC/HAP emissions that continue to pose residual risks
4. Characterize HAPs from indoor products by conducting test house experiments and validating source models
5. Characterize air toxics from waste and fuel gasification processes
6. Develop measurement methods for accurately quantifying HAPs of interest that are found to be important as non-cancer health endpoints are explored
7. Study As speciation/sorbent interactions in waste and metallurgical industries

Exposure

1. Apply receptor modeling techniques to identify the relative source contributions to ambient air toxics concentrations in urban areas and to personal exposures
2. Expedite human exposure model development and address more HAPs
3. Improve neighborhood scale modeling by developing a sub-grid scale model to be used for hot spot type analyses

Health Effects

1. Improve dosimetric methods for airway irritants, e.g., computational fluid dynamic models
2. Identify the reproductive and developmental hazards of exposure to HAPs during gestation, the early post-natal period, and adolescence
3. Develop biomarkers of exposure and effect for the pulmonary, immune, and nervous systems
4. Characterize the toxicity of inhaled metals, focusing on cardiopulmonary and neurological effects
5. Characterize non-genetic susceptibility factors for HAPs, including age and existing disease
6. Determine whether genetic factors play a role in susceptibility to HAPs

Assessment

1. Develop more timely dose-response assessments and update old IRIS database assessments to keep pace with national scale and residual risk assessments
2. Provide more timely fuel/fuel additive analysis of testing results and delisting consultations

Management and control

1. Evaluate risk management strategies, and identify research needs for mitigation of low concentration VOCs
2. Develop an integrated strategy to control the emissions of air toxics due to recycling/combustion/management of discarded consumer electronics equipment

6.0 APG/APM Table

6.1 APGs and APMs for LTG-1

PERFORMANCE GOALS AND MEASURES		YEAR	LAB/ CENTER	Classifi-cation
LTG 1, <u>Reduce Uncertainty in Air Toxics Risk Assessments</u>: By 2010, provide health hazard and exposure methods, data, and models to enable the Program and Regional Offices to reduce uncertainty in risk assessments of acute, chronic, and multi-pathway exposures to air toxics at the national and regional levels, and conduct 3-5 community-level exposure and epidemiology studies to characterize the risk of air toxics at that scale.		**2010**	**ORD**	**N/A**
APG 40 - <u>Health Effects and Aggregate Exposures</u> -Provide measurements, methods, models, information, assessments, and technical support to OAR, regional, state, tribal, and local offices to estimate human health effects and aggregate exposures to hazardous air pollutants in the Air Toxics Research Strategy		**2003**	**ORD**	Internal
APM 176	Identify a model form which describes the relationship between respiratory effects and concentration and duration of exposure of chlorine in humans (pending final human subjects clearance)	2003	NHEERL	Internal
APM 177	Develop a framework and strategy for quantifying exposure-dose-response relationships and health effects for air toxics cancer and non-cancer outcomes through the use of representative air toxic compounds and biologically-based hazardous air pollutant groups	2003	NHEERL	Internal

APM 178	Methods development and validation of oxidative stress measurements as broadly applicable indicators of toxicity	2003	NHEERL	Internal
APM 20	Produce literature-based chemical mechanisms for 33 air toxic compounds, so that OAQPS, the States can, based on the best information available, predict ambient concentrations and chemical fates	2003	NERL	Internal
APM 21	Produce an aggregate population exposure model for air toxics that will assist risk assessors in evaluating uncertainty and variability in air toxics exposure	2003	NERL	Internal
APM 43	Complete External Review Drafts of four dose-response assessments to support NATA and other risk assessments	2003	NCEA	Internal
APM 44	Provide consultation to State and local agencies and others on health and ecological risk assessment and analysis, and ambient monitoring, emissions, and modeling for hazardous air pollutants	2003	NCEA	Internal
APM 45	Provide support to Program Office regulatory actions and rulemakings including listing/delisting assessments, national scale assessments, and residual risk assessments	2003	NCEA	Internal
APM 46	Provide technical evaluation of Alternative II fuel/fuel additive (e.g., oxygenates) toxicity testing results submitted by producers	2003	NCEA	Internal
APM 47	Improve BMD and CATREG software to include other statistical packages and models for improved analysis of cancer and neurotoxicity data	2003	NCEA	Internal

APG 14 - <u>Extrapolation</u> - Develop and evaluate the applicability for extrapolating health effects from route to route and from animals to humans for selected HAP groups in order to enhance the applicability and speed with which health effects information can be applied to risk assessments		**2004**	**ORD**	**Internal**
APM 141	Evaluation of the validity of route-to-route extrapolation for selected air toxics within a HAP group	**2004**	**NHEERL**	**Internal**
APG - <u>Preliminary Acute Approach</u> - Provide data on exposure, health effects, and mode of action of HAPs to support ORD's development of a preliminary approach to acute dose-response and exposure assessment for OAR's use in acute risk assessments		**2005**	**ORD**	**Internal**
APM 179	Characterize the acute health effects of selected inhaled HAPs	2003	NHEERL	Internal
APM 174	Develop and validate a method for studying the effects of toluene exposure on cognitive function in humans	2003	NHEERL	Internal
APM 317	Provide initial linkage of CMAQ model results at neighborhood scales with human exposure models	2004	NERL	Internal
APM	Predict the acute neurotoxicity of untested HAPs in humans based on results of experimental studies in animals	2005	NHEERL	Internal
APM	External Review Draft of revised Acute Health Assessment methodology	2005	NCEA	Internal
APM	Report on application of the acute methodology to assessment of acute (and longer term) toxicity	2005	NCEA	Internal

APM	Report on the feasibility of applying biologically based mechanistic models to extrapolate dose-response from longer term to acute exposures for HAP chemicals	2005	NCEA	Internal
APM	Synthesis document on preliminary acute approach	2005	NCEA	Internal
APG - <u>Feasibility/Pilot Studies</u> - Determine the feasibility of conducting a community-level epidemiological assessment by developing source characterization tools, exposure factors and models, and health endpoint information for use in OAR, Region, state, tribal and local community-level assessments		**2007**	**ORD**	**Internal**
APM	Report on the use of exposure measurements and models to classify exposure in epidemiological studies	2006	NERL	Internal
APM	Determine the health endpoints to quantify in epidemiological studies of the health effects of exposure to air toxics	2007	NHEERL	Internal
APM	Determine the demographic subpopulations with greatest potential susceptibility to health effects from exposure to air toxics	2007	NHEERL	Internal
APM	Identify sources that have a significant impact on a large number of U.S. communities and how best to characterize them	2007	NRMRL	Internal
APM	Synthesis document on feasibility studies for conducting epidemiological assessments	2007	NERL	Internal

APG - <u>Improved Chronic Approach</u> - Provide data on dose-response relationships, mechanisms, biomarkers of effect, and methods of assessment which reduce uncertainty in chronic toxicity and carcinogenicity risk assessments of chronic exposures conducted by OAR		**2008**	**ORD**	**Internal**
APM 180	Determine which route of metabolism leads to the ultimate carcinogenic metabolite of 6-nitrochrysene, a carcinogenic component of diesel emissions	2003	NHEERL	Internal
APM 89	Report submitted to external peer review on a methodology for analysis of nasal dosimetry and delivered dose to the lung to be used in developing RfC values and reducing uncertainty in risk assessment	2004	NCEA	Internal
APM 145	Further explore the relationship between biomarkers of effect and carcinogenic potency for POMs	2004	NHEERL	Internal
APM	Develop proteomics method to determine common mechanisms of injury for representative compounds in classes of halogenated organics, metals/mineral, aldehydes and ketones, and PAHs	2005	NHEERL	Internal
APM	Apply proteomics method to the determination of common mechanisms of injury for specific compounds in classes of metals/mineral and PAHs	2006	NHEERL	Internal
APM	Determine the relative contributions of stable vs. unstable POM-DNA damage in inducing carcinogenesis	2006	NHEERL	Internal
APM	Benchmark Dose Software revised to support dose-response assessment of saturable effects using dichotomous models	2006	NCEA	Internal

APM	Preliminary report on potential applications of genomics/proteomics to improve risk assessment of respiratory toxicology	2006	NCEA	Internal
APM	Apply proteomics method to determine common mechanisms of injury for representative compounds in the HAP classes of halogens and aldehydes and evaluate this method across all classes of HAPs	2007	NHEERL	Internal
APM	Analysis of nasal uptake/lung dose in various subpopulations for chemicals with different reactivities	2007	NCEA	Internal
APM	Improve Benchmark Dose Software through development of modified continuous models which estimate fraction affected in toxicity studies	2007	NCEA	Internal
APM	Report on results, recommendations, and response of workshop on revisions to methodology for assessment of inhalation toxicity (RfC methodology)	2007	NCEA	Internal
APM	Develop new software tools to support model development that reduces the uncertainty in the interpretation of epidemiology data	2008	NCEA	Internal
APM	Prepare draft revised inhalation toxicity methodology for presentation to and review by the Risk Assessment Forum	2008	NCEA	Internal
APM	Report on the use of genomics/proteomics and computational toxicology in risk assessment of respiratory toxicology and HAP chemicals	2008	NCEA	Internal
APM	Synthesis document on improved chronic methodology	2008	NCEA	Internal

APG - <u>Improved Acute Approach</u> -Provide data on human exposures, mode of action, classification of HAPs by toxicity and extrapolation to improve the risk assessment of acute exposures used by OAR and other clients		**2008**	**ORD**	**Internal**
APM	Identify model forms common to animals and humans which relate pulmonary and neurobehavioral effects to exposure and dose for representative HAPs	2005	NHEERL	Internal
APM	Determine the utility of *in vitro* assays to detect the neurotoxicity of VOCs	2005	NHEERL	Internal
APM	Identify mechanistic determinants of uptake and tissue interaction and determine the degree of homology between species using chlorine as a prototype for reactive air toxics	2005	NHEERL	Internal
APM	Acute Reference Exposure (ARE) assessments developed as special case studies for selected key HAP chemicals	2006	NCEA	Internal
APM	Database of HAP chemical data needed to develop biologically based mechanistic models for extrapolation of dose-response from longer term to acute exposures	2006	NCEA	Internal
APM	Apply physiologically based pharmacokinetic (PBPK) models for route-to-route extrapolations to enable development of dose-response relationships for HAPs when data are available only for the oral route	2006	NHEERL	Internal
APM	Determine the potency of various air toxics in the induction of lung inflammation and allergy in humans	2006	NHEERL	Internal
APM	Summary report on internal dose metrics and acute VOC neurotoxicity	2006	NHEERL	Internal

APM	Produce data on short term human exposures to air toxics	2006	NERL	Internal
APM	Identify the critical microenvironments that influence acute human exposures to air toxics and the critical factors which influence these exposures	2007	NERL	Internal
APM	Test generality of mode of action across VOCs, and use information to categorize VOCs in terms of neurotoxicity	2007	NHEERL	Internal
APM	Compare tissue concentrations estimated by a multi-route exposure model to experimentally measured tissue concentrations	2007	NHEERL	Internal
APM	Characterize exposure-dose-response for humans and laboratory animals exposed to chlorine and describe these as functions of concentration, minute ventilation, and duration of exposure	2007	NHEERL	Internal
APM	Complete improvements to categorical regression software and application to acute and longer term assessment of HAP chemicals	2007	NCEA	Internal
APM	Report on scientific characterization of dose-response for respiratory irritants and toxicants (e.g., varying duration and concentration; *in vivo* vs *in vitro* data; severity)	2007	NCEA	Internal
APM	Report on differences in approaches to acute versus chronic dosimetry in the respiratory tract	2007	NCEA	Internal
APM	Case study with a selected HAP chemical demonstrating the use of a biologically based mechanistic model to extrapolate dose-response from longer term to acute duration exposures	2008	NCEA	Internal

APM	Update modeling tools with recent exposure studies and demonstrate its applicability for estimating acute exposures	2008	NERL	Internal
APM	Demonstrate neighborhood scale modeling capabilities of CMAQ to produce more refined temporal variability in ambient concentrations	2008	NERL	Internal
APM	Provide a quantitative extrapolation model of acute solvent behavioral effects in rats and humans	2008	NHEERL	Internal
APM	Establish relationships based on mode of action to support generalizations for cancer and non-cancer outcomes for representative HAPs, and revise HAP classification accordingly	2008	NHEERL	Internal
APM	Determine the influence of model input parameters on model predictions	2008	NHEERL	Internal
APM	Synthesis document on improved acute approach	2008	NCEA	Internal
APG - <u>Cumulative Risks</u> -Collect data and develop methods and models for characterizing and predicting multipollutant, multipathway exposures and the toxicity of HAP mixtures based on mode of action to reduce uncertainty in OAR and other client risk assessments		**2009**	**ORD**	**Internal**
APM	Characterize acute noncancer health effects and develop PBPK models for the binary interactions of trichloroethylene plus carbon tetrachloride and trichloroethylene plus chloroform	2006	NHEERL	Internal
APM	Characterize the neurotoxicity of VOC mixtures based on mode of action at specific neural substrates using in vitro techniques	2007	NHEERL	Internal

APM	Collect data on multipathway exposures to air toxics to support model development	2007	NERL	Internal
APM	Characterize the acute behavioral effects of simultaneous exposure to multiple solvents in rats and humans	2008	NHEERL	Internal
APM	Develop models to estimate exposure and dose to air toxic pollutants with multiple pathways of exposure so that EPA can conduct assessments of cumulative exposure and risks	2009	NERL	Internal
APM	Synthesis document on cumulative risk methods	2009	NHEERL	Internal
APG - <u>Susceptibility</u> - Determine the susceptibility factors associated with exposure and toxicity of HAPs to reduce uncertainty in OAR and other clients' risk assessments		**2009**	**ORD**	**Internal**
APM	Describe effects of metals on animal models of allergic asthma and infection	2005	NHEERL	Internal
APM	Develop a PBPK model for aged rats, using age-appropriate physiological input parameters	2006	NHEERL	Internal
APM	Report on current empirical approaches to age-related alterations in respiratory tract dosimetry and potential application to HAP chemicals	2007	NCEA	Internal
APM	Determine whether short-term exposure to selected hazardous air pollutants results in exacerbation of asthma in humans	2008	NHEERL	Internal
APM	Examine predisposing genetic links to carbonyl sensitivity	2008	NHEERL	Internal
APM	Examine whether sensitivity to classes of air toxics is genetically linked	2009	NHEERL	Internal

APM	Synthesis document on susceptibility to health effects	2009	NHEERL	Internal
APG - <u>Community Assessment Tools</u> - Validate exposure and epidemiology assessment tools to support OAR, Region, state, tribal, and local community-level risk assessments		**2010**	**ORD**	**Internal**
APM	Develop dose-response assessments for cabonyl HAPs and chlorine, using exposure-dose response models, SAR, gene expression, relative potency, and information on genetic susceptibility	2010	NHEERL	Internal
APM	Conduct measurements and apply modeling tools to classify human exposures in multiple community-based epidemiological studies	2010	NERL	Internal
APM	Synthesis document on community assessment tools	2010	NHEERL	Internal

6.2 APGs and APMs for LTG-2 (Italicized APMs are supporting LTG 1 APMs)

PERFORMANCE GOALS AND MEASURES		YEAR	LAB/ CENTER	Classifi-cation
LTG 2, <u>Implement Risk Reduction of Air Toxics</u>: By 2008, produce fifteen new or modified tools in the form of methods, models, or assessments that enable officials at the national, regional, state, or local community level to identify or implement cost-effective approaches to reduce risks from stationary point, area, mobile, or indoor sources of air toxics.		2008	ORD	N/A
APG 41 - <u>Atmospheric and Emission Models</u> - Provide first generation atmospheric and emission models to OAR in order to estimate fate, ambient concentrations, and mobile source emissions of air toxics at a more refined scale		**2003**	**ORD**	**Internal**
APM 150	Produce an upgraded MEASURE modeling system capable of estimating air toxics emissions from mobile sources under various modes of operation at refined spatial scales to support OAR toxic assessments	2003	NRMRL	Internal
APG - <u>Emission Factors from Non-road Mobile Sources</u>: By 2005, provide improved emission factors for air toxics emissions from non-road sources to support OAR implementation of the technical analysis plan (TAP) contained in the 2000 Mobile Air Toxics Rule		**2005**	**ORD**	**Internal**
APM 264	Deliver to OTAQ air toxics emissions data from small engines for use in models needed to improve and update the National Emissions Inventory	2004	NERL	Internal
APM	Characterize emissions from additional small non-road engine type	2005	NERL	Internal
APM	Synthesis document on emissions factors from non-road mobile sources	2005	NERL	Internal

APG - <u>Residual Risk - Second Group</u>: By 2005, transfer to OAR the latest dose-response assessment values, exposure information and tools, and emissions information for use in developing residual risk standards that will be developed between 2006 and 2008		**2005**	**ORD**	**Internal**
APM 92	Develop for external peer review four dose-response assessments intended for the IRIS and which support NATA including residual, urban, mobile source, indoor air or other risk assessments	2004	NCEA	Internal
APM 93	Consultation to support Program Office, regions, states, and tribes in assessments, regulatory actions, and rule makings including technical evaluation of fuel/fuel additive Tier II test data	2004	NCEA	Internal
APM	Report on feasibility of compiling and prioritizing chemical-specific health hazard information for long term support of residual risk assessments	2005	NCEA	Internal
APM	Report on the most recently available exposure information and tools for use in developing residual risk standards	2005	NERL	Internal
APM	*Synthesis document on preliminary acute approach. FROM LTG 1*	*2005*	*NCEA*	*Internal*
APM	Synthesis document on tools for residual risk	2005	NCEA	Internal
APG - <u>NATA Metals and Aldehydes</u>: By 2006, upgrade air quality models; generate source emission factors, models, and new measurement techniques; provide dose-response assessment values and exposure estimates; and evaluate risk management options for metals and aldehydes, particularly those of greatest concern in urban areas		**2006**	**ORD**	**Internal**

APM 216	Report on the addition of next set of air toxics chemicals to the CMAQ's modeling ability for use in air toxics assessments	2004	NERL	Internal
APM	Characterize atmospheric chemistry for aldehydes and metals on the Urban Air Toxics list	2006	NERL	Internal
APM	Update CMAQ modeling system to include aldehydes and metals on the Urban Air Toxics list	2006	NERL	Internal
APM	Evaluate a refined air quality model to provide mercury deposition to water and terrestrial surfaces	2006	NERL	Internal
APM	Provide exposure and dose modeling tools for use in conducting exposure assessments for priority aldehydes and metals	2006	NERL	Internal
APM	Evaluate metal speciation of arsenic, nickel, and chromium in selected combustion systems	2006	NRMRL	Internal
APM	*Synthesis document on preliminary acute approach. FROM LTG 1*	*2005*	*NCEA*	*Internal*
APM	Synthesis document on metals and aldehydes information for use in NATA	2006	NRMRL	Internal
APG - <u>Indoor Air Toxics Exposure and Emissions</u>: By 2008, provide improved data on emissions, pollutant formation and transformation, and exposure estimates for priority indoor air toxic pollutants, particularly aldehydes, and identify cost effective approaches to reduce human exposure		**2008**	**ORD**	**Internal**

APM	Complete development of a model to predict how infiltration of priority outdoor toxics impacts indoor concentrations and determine how to link this model with the indoor toxics source emissions model	2005	NRMRL	Internal
APM	Assemble stakeholder group and identify candidate indoor sources and mitigation technologies for risk management evaluation	2005	NRMRL	Internal
APM	Develop risk- and exposure-based criteria for performance evaluations for indoor air mitigation technologies	2006	NRMRL NERL NHEERL	Internal
APM	Generate data and conduct analyses to identify the relationships between ambient air toxics, indoor concentrations and actual human exposures and the factors which influence these relationships	2006	NERL	Internal
APM	Perform risk management evaluation, reporting on ranking of options for indoor air mitigation technologies	2007	NRMRL	Internal
APM	Test and validate selected options for indoor air mitigation technologies	2008	NRMRL	Internal
APM	Synthesis document on indoor air toxics exposure and emissions	2008	NRMRL	Internal
APM	Apply modeling tools to identify the critical indoor microenvironments of concern and the factors which influence human exposure to air toxics in these microenvironments	2008	NERL	Internal
APM	**Synthesis document on indoor air toxics exposure and emissions**	**2008**	**NRMRL**	**Internal**

APG - <u>Residual Risks -Third Group</u>: By 2007, transfer to OAR the latest dose-response assessment values, exposure information and tools, and emissions information for use in developing residual risk standards that will be developed between 2008 and 2010		**2007**	**ORD**	**Internal**
APM	Develop for external peer review four dose-response assessment which support residual risk assessments to be conducted in 2008-2010	2005	NCEA	Internal
APM	Develop for external peer review four dose-response assessment which support residual risk assessments to be conducted in 2008-2010	2006	NCEA	Internal
APM	Develop for external peer review four dose-response assessment which support residual risk assessments to be conducted in 2008-2010	2007	NCEA	Internal
APM	Report on the most recently available exposure information and tools for use in developing residual risk standards	2007	NERL	Internal
APM	*Synthesis document on preliminary acute approach. FROM LTG 1*	*2005*	*NCEA*	*Internal*
APM	Synthesis document on tools for residual risk	2007	NCEA	Internal
APG - <u>NATA Halides and PAHs</u>: By 2009, upgrade air quality models; generate source emission factors, models and new measurement techniques; provide dose-response assessment values and exposure estimates; and identify risk management options for halides and PAHs, particularly those of greatest concern in urban areas		**2009**	**ORD**	**Internal**

APM 116	Report on air toxics from uncontrolled burning of forest and agricultural biomass based on in-house and field sampling (conducted by others) to support upgrades of AP-42 and used in air quality models	2004	NRMRL	Internal
APM	Deliver a comprehensive report of air toxics emitted from uncontrolled burning or residential/construction waste based on in-house and field sampling (conducted by others) to support upgrades of AP-42 and for use in air quality models	2006	NRMRL	Internal
APM	Develop, evaluate, and field-test the Jet-REMPI technology for measurement of trace organics	2007	NRMRL	Internal
APM	Validate the Jet-REMPI method through testing multiple source types and through comparison with other measurement methods/standards and produce guidance for use of this method	2008	NRMRL	Internal
APM	Characterize atmospheric chemistry for Halides and PAHs on the Urban Air Toxics list	2008	NERL	Internal
APM	Deliver an updated SHEDs model to OAR which predicts exposures to priority halides and PAHs	2008	NERL	Internal
APM	Update CMAQ modeling system to include Halides and PAHs on the Urban Air Toxics list	2009	NERL	Internal
APM	*Synthesis document on improved acute approach. FROM LTG 1*	*2008*	*NCEA*	*Internal*
APM	*Synthesis document on improved chronic approach. FROM LTG 1*	*2008*	*NCEA*	*Internal*
APM	*Synthesis document on cumulative risk methods. FROM LTG 1*	*2009*	*NHEERL*	*Internal*

APM	Synthesis document on halides and PAH information for use in NATA	2009	NERL	Internal
APG - <u>Assess Need for Additional Regulatory Options for Mobile Source Air Toxics</u>: By 2008, improve estimates of air toxic exposures in mobile source microenvironments and develop IRIS values and comparative evaluations to assist OAR in determining whether additional mobile source regulatory controls are needed to protect public health		**2008**	**ORD**	**Internal**
APM 224	Enhance current human exposure models for air toxics to allow risk assessors to better address exposures related to mobile sources	2004	NERL	Internal
APM	Complete a paper on recent testing of air toxic emissions from heavy-duty diesel trucks	2005	NRMRL	Internal
APM	Complete development of enhanced methods for temporal and spatial allocation of truck activity in the modal-based MEASURE model	2005	NRMRL	Internal
APM	Report on comparative evaluations of air toxics and multi-media environmental and health impacts of key fuel options/choices over the full life-cycle	2006	NCEA	Internal
AM	Complete an operational MEASURE model for estimating mobile source air toxics emissions in a GIS environment with improved spatial and temporal allocation of vehicle activity and emission factors	2007	NRMRL	Internal
APM	Using measurement data, identify relationships between human exposures to air toxics and mobile source emissions and the critical factors which influence these relationships	2007	NERL	Internal

APM	Apply modeling tools to characterize the range of population exposures related to mobile sources and identify the critical related microenvironmental factors	2008	NERL	Internal
APM	Synthesis document on regulatory options for mobile sources	2008	NERL	Internal

7.0 Notes

When comparing the existing APGs and APMs in the Integrated Resource Management System (IRMS) with this MYP, the following changes should be made to IRMS in order to make the two compilations of research activities consistent:

1. Delete FY 2007 APG 73 and move associated FY 2004 APM 89 to FY 2008 APG "Improve Chronic"

2. Delete FY 2005 APG 77 and move associated FY 2004 APM 92 and APM 93 to FY 2005 APG "Residual Risk–2nd Group"

3. Delete FY 2005 APG77 and move associated FY 2004 APM 116 to FY2009 APG "Halides and PAHs"

4. Delete FY 2006 APG 71 and move associated FY 2004 APM 317 to FY 2005 APG "Preliminary Acute Approach"

5. Delete FY 2006 APG 71 and move associated FY 2004 APM 216 to FY 2008 APG "NATA Aldehydes and Metals"

6. Delete FY 2008 APG 74 and move associated FY 2004 APM 224 to FY 2008 APG "Assess Need for Additional Regulatory Options for Mobile Source Air Toxics"

7. Delete FY 2005 APG 79 and move associated FY 2004 APM 264 to FY 2005 APG "Emission Factors from Non-road Mobile Sources"

8. Move APMs 174 and 179 from FY 2003 APG 40 to FY 2004 APG "Preliminary Acute Approach"

Appendix A - Table A-1

List of Urban, Stationary, Mobile, and Indoor Priority Air Toxics and Status of Dose-Response Assessment (IRIS) Development

HAP	URE	RfC	RfC UF	RfD	Status[a]	ATRS Group[b]	Urban Sources[c]	Stationary Sources[d]	Mobile Sources	Indoor Air Sources
Acetaldehyde	X	X	1000		L,D,I,X	AK	X	X	X	X
Acrolein		X	1000		L,D,I,X,	AK	X	X	X	
Acrylonitrile	X	X	1000				X	X		
Aldrin	X			X		H				X
Antimony				X	L,D	M		X		
Arsenic & cmpds	X			X		M	X	X	X	X
Benzene	X				L,D,I,X,	POM/	X	X	X	X
Beryllium & cmpds	X	X	10	X		M	X			
alpha-BHC						H				X
gamma-BHC						H				X
1,3-Butadiene	X				L,D,I,X,	POM/	X	X	X	
Cadmium & cmpds	X				L,D,I,X,	M	X	X		
Carbon tetrachloride	X			X	L,D,I	H	X			X
Chlordane	X	X	1000	X		H				X
Chlorine				X		H		X		
Chloroform	X			X	L,D	H	X	X		X
Chloroprene					L,D,I,X	H		X		
Chromium VI & cmpd (mist,	X	X	90; 300	X		M	X	X	X	
Cobalt						M		X		
Coke oven emissions (PAHs:	X				L,D	POM/	X	X		
Cresol								X		
Cumene	X	X	1000	X		POM/		X		
Dibenzofurans	–					POM/		X		
Dibutylphthalate	–			X	L,D			X		
Dichloroethyl ether	--			X				X		
1,4-Dichlorobenzene		X	100		L,D	H				X
1,3-Dichloropropene	X	X	30	X		H	X			
Dichlorvos	X[O]	X	100	X		H				X
Dieldrin	X			X		H				X
Diesel PM + OG		X	30		L,D,I,X	POM/			X	
1,4-Dioxane	X[o]							X		
Dioxin					L,D,I,X	H	X	X	X	
Epichlorohydrin	X	X	300			H		X		
Ethanol					L,D				X	

HAP	URE	RfC	RfC UF	RfD	Status[a]	ATRS Group[b]	Urban Sources[c]	Stationary Sources[d]	Mobile Sources	Indoor Air Sources
Ethyl benzene		X	300	X		POM/			X	
Ethylene dibromide	X				L,D,I,X	H	X	X		
Ethylene dichloride (1,2-	X			X	L,D	H	X	X		
Ethylene glycol				X				X		
Ethylene oxide					L,D		X	X		
Formaldehyde	X			X	L,D	A/K	X	X	X	X
Gasoline particulate matter									X	
Glycol Ethers [EGBE]		[X]	[30]	[X]				X		
Hexachlorobenzene	X			X		H	X			
Heptachlor	X			X		H				X
n-Hexane		X	300			POM/		X	X	
Hydrazine	X						X			
Hydrochloric acid		X	300					X		
Hydrocyanic acid		X	1000	X				X		
Lead & cmpds						M	X	X	X	
Manganese & cmpnds		X	1000	X		M	X	X	X	
Mercury & cmpds [MeHg]	–	X	30	[X]		M	X	X	X	
Methanol				X	L,D			X		
Methylene chloride	X			X		H	X	X		X
Methyl isobutyl ketone					L,D,I,X,	A/K		X		
Methyl chloride	–	X	1000	--	L,D,I,X,	H		X		X
Methyl ethyl ketone	--	X	1000	X	L,D,I	A/K		X		
Methyl tertiary butyl ether		X	100		L,D,I				X	
Naphthalene	–	X	3000	X	L	POM/		X	X	
Nickel & cmpds	X			X	L,D,I,X	M	X	X	X	
2-Nitropropane		X	1000					X		
Perchloroethylene				X	L,D,I	H	X	X		X
Phenol	--			X				X		
Polychlorinated biphenyls	X				L,D	H	X			
Polycyclic organic matter					L	POM/	X		X	
Propylene dichloride		X	300			H	X			
Quinoline	X[o]	–		–	L,D,I,X,		X			
Styrene		X	30	X	L,D	POM/		X	X	
1,1,2,2-Tetrachloroethane	X					H	X			
Toluene	–	X	300	X	L,D,I,X	POM/		X	X	
Toluene diisocyanate		X	30					X		
1,1,1-Trichloroethane	–				L,D	H		X		
1,1,2-Trichloroethane	X			X		H		X		
Trichloroethylene					L,D,I,X	H	X	X		X

HAP	URE	RfC	RfC UF	RfD	Status[a]	ATRS Group[b]	Urban Sources[c]	Stationary Sources[d]	Mobile Sources	Indoor Air Sources
Vinyl acetate		X	30		L,D			X		
Vinyl chloride	X	X	30	X		H	X	X		
Xylene	–			X	L,D,I,X,	POM/		X	X	

[a]Status of IRIS file development which proceeds in the following order: literature review (L), file development (D), internal review (I), external review (X), consensus review (C), and final (F)
[b]AK = Aldehydes and Ketones/Acylating Agents Group; M = Metals/Minerals Group; POM/HC = Polycyclic Organic Matter/Hydrocarbons Group; H = Halides Group.
[C]Originally selected as urban HAPs.
[d]Stationary Source HAPs designated by associated ACT Standard bin in which they are particularly prevalent. All 188 HAPs identified in the CAAA will be addressed by the residual risk program..
[O]Oral value only; "–" not applicable (RfD) or not classifiable (URE)